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Are you surprised to hear this? Longitudinal spectral speech exposure in older compared to middle-aged normal hearing adults

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Keywords: aging, auditory learning, cognitive hearing, longitudinal plasticity, speech processing

Abstract

Cognitive abilities such as attention or working memory can support older adults during speech perception. However, cognitive abilities as well as speech perception decline with age, leading to the expenditure of effort during speech processing. This longitudinal study therefore investigated age-related differences in electrophysiological processes during speech discrimination and assessed the extent of enhancement to such cognitive auditory processes through repeated auditory exposure. For that purpose, accuracy and reaction time were compared between 13 older adults (62–76 years) and 15 middle-aged (28–52 years) controls in an active oddball paradigm which was administered at three consecutive measurement time points at an interval of 2 wk, while EEG was recorded. As a standard stimulus, the nonsense syllable /ˈa:ʃa/was used, while the nonsense syllable /ˈa:sa/ and a morphing between /ˈa:ʃa/ and /ˈa:sa/ served as deviants. N2b and P3b ERP responses were evaluated as a function of age, deviant, and measurement time point using a data-driven topographical microstate analysis. From middle age to old age, age-related decline in attentive perception (as reflected in the N2b-related microstates) and in memory updating and attentional processes (as reflected in the P3b-related microstates) was found, as indicated by both lower neural responses and later onsets of the respective cortical networks, and in age-related changes in frontal activation during attentional stimulus processing. Importantly, N2b- and P3b-related microstates changed as a function of repeated stimulus exposure in both groups. This research therefore suggests that experience with auditory stimuli can support auditory neurocognitive processes in normal hearing adults into advanced age.

Introduction

Many hearing impaired older adults complain that they get increasingly tired during spoken language comprehension, especially in noisy environments such as restaurants. Thus, it requires substantial cognitive effort to understand a speech signal that is distorted due to hearing loss (McCoy *et al.*, 2005; Stewart & Wingfield, 2009). However, aging per se, independent of hearing loss, reduces cognitive capacity (Salthouse, 1996; Park & Reuter-Lorenz, 2009) because older individuals with normal hearing – as signaled by low pure-tone thresholds – report that they too have problems with speech processing (Pichora-Fuller & Souza, 2003; Hopkins & Moore, 2011; Füllgrabe, 2013; Moore *et al.*, 2014; Füllgrabe *et al.*, 2015). Pure-tone thresholds, as measured by audiogram, are currently the most common parameters to describe hearing loss (Pickles, 2012), even though they are not indicators of cognitive hearing problems (Moore *et al.*, 2014; Giroud *et al.*, in press). Thus, these findings point to the relevance of cognition in speech processing, even in older adults who are labeled as normal hearing according to their audiograms. Speech processing in older adults is therefore not only a function of the auditory periphery and the central auditory circuits, but also cognition (Humes *et al.*, 2012).

Some studies have already shown that treatments such as hearing aids can facilitate cognitive-related speech processing in the hearing impaired (Giroud *et al.*, 2017a); for example, by showing that

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performance in working memory (Doherty & Desjardins, 2015) or performance in dichotic listening tasks (Lavie *et al.*, 2015) was higher in aided compared to unaided listening situations. It is likely that these benefits in cognitive-related auditory tasks result from a facilitation of perceptual auditory processes during the use of hearing aids. Furthermore, it is of the utmost importance to study the extent to which these cognitive resources can be supported by training in normal hearing adults, as already suggested in a previous paper (Giroud *et al.*, 2017b). To this end, we compared normal hearing older adults to a middle-aged control group during spectral speech processing in a repeated measurement design that allowed for the evaluation of longitudinal changes in auditory neurocognitive processing as a function of repeated auditory exposure. At three consecutive measurement time points at two-week intervals, we therefore assessed detection accuracy (ACC) and detection reaction time (RT) of spectrally manipulated syllables in an active oddball paradigm, while EEG was recorded.

The active oddball paradigm is a convenient and practical method for measuring electrophysiological correlates of auditory neurocognitive functions during speech processing. More explicitly, we evaluated the mentioned cognitive auditory processes using the onset and the mean global field power (GFP) of the event-related potentials (ERP) evoked by the active oddball paradigm, namely the N2b and the P3b. In the following, we compare the onset and the mean GFP from the current investigation with the traditionally reported peak amplitude and peak latency, respectively, from previous studies. The N2b component belongs to the N2 ERP family and has previously been related to perceptual auditory processes that occur when a deviant stimulus in a continuous stimulus stream is attended and thereby perceptually categorized as a deviant (Simson *et al.*, 1977; Näätänen & Gaillard, 1983). The P3b component, which belongs to the P300 ERP family, peaks at around 300 ms after stimulus deviation onset and has been described as a neural marker for the cognitive part of speech processing (Kok, 1997; Debener *et al.*, 2002; Polich, 2007; Volpe *et al.*, 2007). As such, it is related to attention, memory processing, and memory updating connected to the processing of the unpredicted deviant stimulus (Kok, 1997; Debener *et al.*, 2002; Polich, 2007; Volpe *et al.*, 2007). Thus, even though the two ERP components, the N2b and the P3b, reflect neural auditory-cognitive processing, we consider the N2b to be linked to attentive perception, whereas the P3b is linked to cognition specifically to attention and memory updating.

Previous cross-sectional research on tone frequency discrimination has shown that older adults had longer N2 and P300 latencies and smaller N2 and P300 amplitudes than younger adults (Bertoli *et al.*, 2005; Schiff *et al.*, 2008; Juckel *et al.*, 2012). Furthermore, age-related differences were stronger for the N2 and the P300 than for earlier ERPs, such as the mismatch negativity (MMN) or the N1, suggesting that age modulated the cognitive part of hearing more strongly than the early perceptual part (Schiff *et al.*, 2008). The combination of fMRI and EEG further revealed that the smaller amplitudes of the P300 in older adults were related to lower BOLD-related responses in frontal (anterior cingulate), temporo-parietal, and subcortical brain regions (hippocampus and amygdala) (Juckel *et al.*, 2012). Because these regions are related to the attentional system, these results suggest that it is more difficult for older adults to focus the attention on acoustic spectral deviations in tones than it is for younger adults (Juckel *et al.*, 2012).

In the current study, we combine a novel longitudinal design with the auditory presentation of syllables (instead of tones as used in previous research) to assess spectral speech processing rather than simple auditory tone perception. Moreover, the vowel–consonant–vowel syllables used in this research incorporated high-pitched fricatives because the processing of high frequencies especially declines relatively rapidly with age (Humes *et al.*, 2012).

We expected to find better behavioral performance (higher ACC and lower RT) for deviants with stronger acoustic deviation to the standard stimulus than lower acoustic deviation (Johnson, 1986; Kok, 1997; Katayama & Polich, 1998; Gaál *et al.*, 2007; Giroud *et al.*, 2017a,b). Furthermore, we expected the better behavioral detection of the deviant with strong acoustic deviation to be reflected in higher mean GFP and shorter onsets of the N2b and P3b components (Johnson, 1986; Kok, 1997; Katayama & Polich, 1998; Gaál *et al.*, 2007; Giroud *et al.*, 2017a,b). We also hypothesized that older adults would evoke lower N2b and P3b mean GFPs and shorter onsets, as has been found in previous studies which compared older and younger adults (Schiff *et al.*, 2008; Juckel *et al.*, 2012). In addition, we expected to find more frontal activation generally on the topography in the older age group during spectral processing (Davis *et al.*, 2008), which would be indicative of a dedifferentiation process associated with age (Cabeza, 2002). We also assumed to find longitudinal changes in that mean GFP would increase and the onset decrease in both groups as a function of auditory neurocognitive learning (Tremblay *et al.*, 1998).

Materials and methods

Participants

Two age groups were compared for this study. Fifteen middle-aged adults (mean age = 40.6 years, SD = 7.4, 8 female) with an age range from 28 to 52 years formed the middle-aged group (MA), while 13 older adults (mean age = 69.23, SD = 3.94, age range 62 to 76, 5 female) were recruited for the older adults group (OA). We only included individuals with pure-tone thresholds < 30 dB for 0.5, 1, 2, 3, and 4 kHz; < 50 dB for 6 kHz; < 60 dB for 8 kHz in the OA group. All participants but five were right handed (four in the OA group and one in the MA group), as indicated by standard handedness questionnaires (Annett, 1970; Bryden, 1977). All participants were native German or Swiss German speakers and none of them reported any history of past or present neurological, psychiatric, or neuropsychological disorders. In addition, they reported not suffering from chronic tinnitus and denied the consumption of drugs or illegal medication.

Written informed consent was obtained from all participants, and the local ethics committee of the University of Zurich approved the study. Participants were compensated for their participation.

Hearing

To assess the hearing performance of the two groups, the online digit triplets test (Buschermöhle *et al.*, 2014, 2015) was performed. During the test, participants were asked to enter the digits they had heard into the computer. The digits were presented with fixed noise, while the triplet's level varied adaptively to locate the 50% intelligibility threshold of the triplets. All participants had lower signal-to-noise ratio (SNR) than 2.9 dB, which has been previously classified as a 'good' SNR (Smits *et al.*, 2006) and therefore normal hearing.

Stimuli

The nonsense syllables (logatomes) asa (/ˈa:sa/), ascha (/ˈa:ʃa/), and afa (/'a:fa/) from the phoneme perception test (Boretzki *et al.*, 2011; Schmitt *et al.*, 2015) were used in our study. This stimulus material had been established previously in other studies using EEG (Giroud

et al., 2017a,b). The logatomes /ˈa:ʃa/ and /ˈa:sa/, /'a:fa/ and /ˈa:sa/, and /ˈa:ʃa/ and /'a:fa/, respectively, were morphed together to create two equidistant intermediate stimuli (Zorn, 2000). In this study, we used the EEG data evoked by the stimulus pair ascha (/ˈa:ʃa/) and asa (/ˈa:sa/) and its two morphing steps. Together these four logatomes were presented in one stimulus block. The logatome /ˈa:ʃa/ was presented as the standard stimulus; the morphed stimulus with the slighter acoustic deviation from the standard was used as Deviant 1 (difficult); the morphed stimulus with the higher acoustic deviation from the standard was used as Deviant 2 (moderate); and the logatome /ˈa:sa/ was called Deviant 3 (easy).

Experimental procedure

The participants were invited for three EEG sessions in total. After the first recording (T1), they were retested 2 wk (T2) and 4 wk (T3) after T1. Each T was scheduled at the same time of day to control for changes in attention during the day.

At each EEG session, participants were seated in a comfortable chair at a distance of about 75 cm in front of a speaker which was placed in front of a screen. Stimulus material was presented at 65dB SPL with some exceptions: Participants were asked to set the volume level of the /ˈa:ʃa/ and the /ˈa:sa/ logatome to an equal loudness level and, if the volume was not set to the same level by the participants, a jitter in volume for the standard stimulus was introduced. A jitter of 1 dB was used if the difference between the two stimuli was set to 1 dB, or a jitter of 2 dB if the difference between the two stimuli was set to 2 dB or more. This procedure allowed participants to detect a deviant only by its perceived qualitative difference to the standard rather than by its perceived difference in loudness. During EEG testing, participants were instructed to fixate on the cross presented on the screen in order to avoid eye movement artifacts. Presentation software (www.neurobs.com; version 14.5) controlled the experiment and presented the standard stimulus 540 times ($P = 0.75$), while each deviant was presented 60 times ($P = 0.083$) in a randomized order with an inter-stimulus interval of 730 ms. Participants were asked to listen to the stream of stimuli and to press the mouse button with the right index finger when a deviant stimulus was identified. Only correctly identified deviants were averaged, resulting in a maximum of 60 trials per deviant and 540 trials for the standard stimulus. This experimental procedure had already been tested in a previous study (Giroud *et al.*, 2017b).

EEG recordings and preprocessing

EEG was continuously recorded using the high-density Geodesic EEG system (Electrical Geodesics, Inc., USA) with 256 scalp electrodes. Impedances were kept below 30 kΩ. The data was online band-pass filtered between 0.1 and 100 Hz and electrode Cz served as the online reference. The data was recorded with a sampling rate of 500 Hz. Offline, the data was re-referenced to linked mastoids for visual inspection of the grand averages using three electrode pools along the midline (Schiff *et al.*, 2008) (frontal: E14, E15, E16, E22, E23, E6, E7; central: Cz, E132, E186, E45, E81, E9; parietal: E100, E110, E119, E128, E129, E130, E89), and afterwards to average references for further data analyses. For the preprocessing steps, Brain Vision Analyzer Software (Version 2.0.4, Brainproducts, Munich, Germany) was used. After removing electrodes placed on the cheeks and on the neck, the data was band-pass filtered between 0.1 and 20 Hz (24 dB/oct). An independent component analysis (ICA) was applied to remove eye movements and eye blinks (Jung *et al.*, 2000). Noisy channels were interpolated (Perrin *et al.*, 1987), and movement artifacts were removed with a semi-automatic raw data inspection. After this, the continuous data was segmented into 1300-ms epochs (from 100 ms pre-stimulus to 1200 ms post-stimulus) and the baseline was corrected relative to the 100 to 0 ms pre-stimulus time period. Six participants only detected less than 33% of the difficult deviants, which resulted in a low number of EEG trials to analyze for these individuals (below 20 trials) and therefore not allowing for a reliable ERP calculation for the difficult deviant. To avoid completely excluding these participants, we rather excluded this condition, Deviant 1 (difficult), in the following analyses. Thus, for each participant, we created averages for the standard stimulus, for the moderate Deviant 2 and for the easy Deviant 3, separately for each T.

Microstate analysis

We used microstate analysis to investigate ERPs in a data-driven and topographical manner (Giroud *et al.*, 2017a,b). This method can be used for evaluating temporally stable topographical configurations, the so-called microstates, measured with high-density EEG (Pascual-Marqui *et al.*, 1995), which can then be compared between groups and conditions. Furthermore, microstates have been shown to correspond to functionally relevant periods that are temporally and spatially related to the ERP components (Michel *et al.*, 2009). For example, they can be compared statistically between groups and conditions using their mean GFP and onset. The mean GFP is defined as the standard deviation of the potentials at all electrodes of an average reference map averaged for one microstate (Skrandies, 1990); the onset is defined as the time point of the first assignment of the grand average ERP signal to one map (Murray *et al.*, 2008). We computed a microstate analysis for the ERP time interval starting from −100–1200 ms after stimulus onset. Using a *k*-means algorithm, the grand averages of all conditions were clustered into stable time periods with Ragu software (version of 20. Jan 2015) operating on MATAB 2012b (The MathWorks Inc., Natick, MA, USA) (Koenig *et al.*, 2011). To find the optimal number of microstates between 3 and 35, a cross-validation algorithm was used, as implemented in Ragu (Koenig *et al.*, 2011, 2014). Fifty random initializations for each number of microstates between 3 and 35 were calculated, while the cross-validation procedure was applied to each of these 50 times. In addition, briefly occurring microstates were excluded by applying a 'smoothing' with 10 points and a penalty factor of 3. After this, the grand averaged data of each condition STIM (standard, moderate deviant, easy deviant), T (T1, T2, T3), and group (MA, OA) were separately fitted back to the clustered microstates using randomization statistics (Koenig & Melie-García, 2009; Koenig *et al.*, 2011). First, the variances between the factors STIM (standard, moderate deviant, easy deviant), T (T1, T2, T3), and group (MA, OA) were computed for each effect of interest (onset and mean GFP). Then, the data was shuffled repeatedly with 1000 repetitions (to obtain an α-level of 0.05). For each repetition, the variance of all effects of interest was computed, while the comparison between the distribution of the real variance and the distribution of the shuffled data was used to obtain the probability of the observed difference between the STIM, T, and group factors, and their compatibility with the null hypothesis. In this way, we obtained p-values for the main effect T (T1, T2, T3), for the main effect STIM (standard, moderate deviant, easy deviant), for the main effect age group (MA, OA), and for the interactions between these factors separately for the onset and the mean GFP of each microstate. Because this procedure analyzes microstates of grand averaged data, no single subject information on microstates can be obtained.

Thus, it is not possible to calculate correlations with behavioral data and the microstate markers.

Analysis of behavioral data

The ACC of the deviant detection and the mean RT for correct trials were computed for each of the two deviants, for each T, separately for each subject. Afterwards, a 2 * 3 * 2 (*STIM*: easy deviant, moderate deviant; *T*: T1, T2, T3; *group*: OA, MA) repeated-measures ANOVA was calculated, first for ACC, then for RT. The ANOVAS were followed by pairwise *t*-tests corrected for multiple comparisons applying Bonferroni's correction, when appropriate. The alpha level for all statistical analyses was set to $\alpha = 0.05$. Effect sizes were indicated by partial eta-squares (η^2_p).

Results

Behavioral performance

The 2 * 3 * 2 (*STIM*: easy deviant, moderate deviant; *time point T*: T1, T2, T3; *group*: OA, MA) repeated-measures ANOVA for ACC revealed a main effect of STIM ($F_{1,26} = 6.63$, $P = .02$, $\eta^2_p = .20$), a main effect group ($F_{1,26} = 8.80$, $P = .006$, $\eta^2_p = .25$), and an interaction between STIM and group ($F_{1,26} = 4.89$, $P = .04$, $\eta^2_p = .16$). The analysis showed that the ACC for the easy deviant was higher than for the moderate deviant, as expected. Furthermore, and also as expected, the middle-aged adults detected the deviants with higher accuracy than older adults, especially the more difficult deviant. The analysis did not reveal a main effect of *T* ($F_{2,52} = 2.28$, $P = .113$, $\eta^2_p = .08$), nor an interaction between T * group ($F_{2,52} = 2.69$, $P = .078$, $\eta^2_p = .09$), nor an interaction between T * STIM ($F_{2,52} = 1.96$, $P = .151$, $\eta^2_p = .07$), nor an interaction between T * STIM * group ($F_{2,52} = .93$, $P = .400$, $\eta^2_p = .04$).

The same repeated-measures ANOVA for RT resulted in a main effect of *T* ($F_{2,52} = 9.00$, $P < .001$, $\eta^2_p = .26$). The post hoc *t*-tests showed that from T1 to T2 ($P = .01$) and T3 ($P = .004$), there was a decrease in RT. Furthermore, there was a main effect of STIM ($F_{1,26} = 27.79$, $P < .001$, $\eta^2_p = .52$) and an interaction between STIM and group ($F_{1,26} = 9.49$, $P = .01$, $\eta^2_p = .27$), revealing that the easier deviant was detected faster than the moderate deviant and that this difference was larger in OA compared to MA (see Fig. 1 for behavioral data). The analysis did not reveal a significant interaction between T * group ($F_{2,52} = .21$, $P = .815$, $\eta^2_p = .01$), nor between T * STIM ($F_{2,52} = 2.73$, $P = .074$, $\eta^2_p = .10$), nor between T * STIM * group ($F_{2,52} = .18$, $P = .833$, $\eta^2_p = .01$).

Thus, as expected, we found higher and faster detection accuracy for the deviants with more acoustic difference to the standard stimulus. In addition, and also as hypothesized, the older adults performed with lower accuracy than the middle-aged adults in the oddball detection task, especially when the acoustic difference between deviant and standard was small. These age-related differences in accuracy reflect the expected decline in spectral speech processing with age. However, repeated stimulus exposure can lead to decreases in deviant detection reaction times in both age groups.

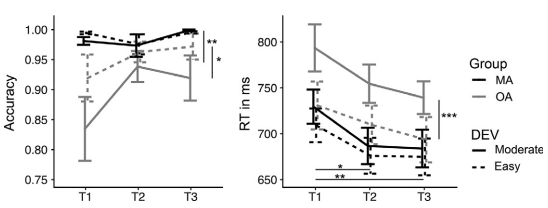

FIG. 1. The behavioral data across the three measurement time points (T1, T2, T3) with the accuracy of deviant detection on the left side and the reaction times on the right side. MA, middle-aged controls; OA, older adults; moderate deviant, deviant with weaker acoustic deviation; easy deviant, deviant with stronger acoustic deviation. Error bars indicate standard errors. *$P < .05$, **$P < .01$, ***$P < .001$.

Microstate analysis

For visual inspection, Fig. 2 depicts the ERPs derived from three different electrode pools.

The microstate analysis on the STIM and T within-subject factors and group as between-subject factor yielded five representative topographic scalp maps (Microstate 1–5), meaning that the maximum grand average correlation was reached when using these five microstate classes. These are depicted in Fig. 3A and the associated time courses of the GFP are depicted in Fig. 3B. As can be seen in Fig. 3B, a posterior and a central N2b (Microstate 1 and 2, respectively) are followed by a frontal, a central, and a posterior P3b (Microstate 3, 4, and 5, respectively) which were evoked by the spectral acoustic deviation starting from 240 ms after stimulus onset. To exclude possible confounding effects in the back-fitting procedure (because microstates can occur in the chosen time windows repeatedly, representing ERPs other than the N2b or the P3b, e.g. the posterior N2b which also occurs more than 1000 ms after stimulus onset), a priori time windows were defined for the microstates' back fitting. Both the posterior N2b (Microstate 1) and the central N2b (Microstate 2) were fitted back for the interval between 240 and 750 ms after stimulus onset, the frontal P3b (Microstate 3) was fitted back for the time interval between 400 and 1100 ms, and the central P3b (Microstate 4) was fitted back for the interval between 500 and 1200 ms. In the following, the mean GFP and the onset of each of these five microstates will be described as a function of T, STIM, and group (see also Fig. 4), thus disentangling the longitudinal modulations occurring after repeated exposure, and age-related differences of perceptual and attentional neurocognitive processes, as evoked by the weaker and stronger acoustic deviations.

Microstate 1: posterior N2b

For the onset of Microstate 1, we found a main effect group ($P = .008$), showing that the onset of Microstate 1 was earlier in the MA group (272 ms) than in the OA group (338 ms). Furthermore, there was a main effect of STIM ($P = .018$), revealing that the onset of Microstate 1 was earlier when evoked by the easy deviant (298 ms) as compared to the moderate deviant (306 ms) and the standard stimulus (334 ms). However, there was no main effect T ($P = .056$), no interaction between T * group ($P = .139$), no interaction between STIM * group ($P = .448$), and no interaction between T * STIM ($P = .132$). There was a T*STIM*group interaction ($P = 0.003$).

The mean GFP of Microstate 1 changed as a function of STIM ($P < .001$), showing that the mean GFP of Microstate 1 evoked by the two deviants (moderate deviant: 0.57 μV, easy deviant: 0.40 μV) was higher compared to the standard stimulus (0.17 μV). There was no main effect of group ($P = .104$), nor T ($P = .319$), nor was there an interaction between T * group ($P = .452$). However, we found an interaction between STIM * group ($P < .001$), showing that the mean GFP of Microstate 1 was smaller in OA than in MA when evoked by the moderate deviant (OA: 0.32 μV, MA: 0.69 μV), but not different when evoked by the easy deviant (OA:

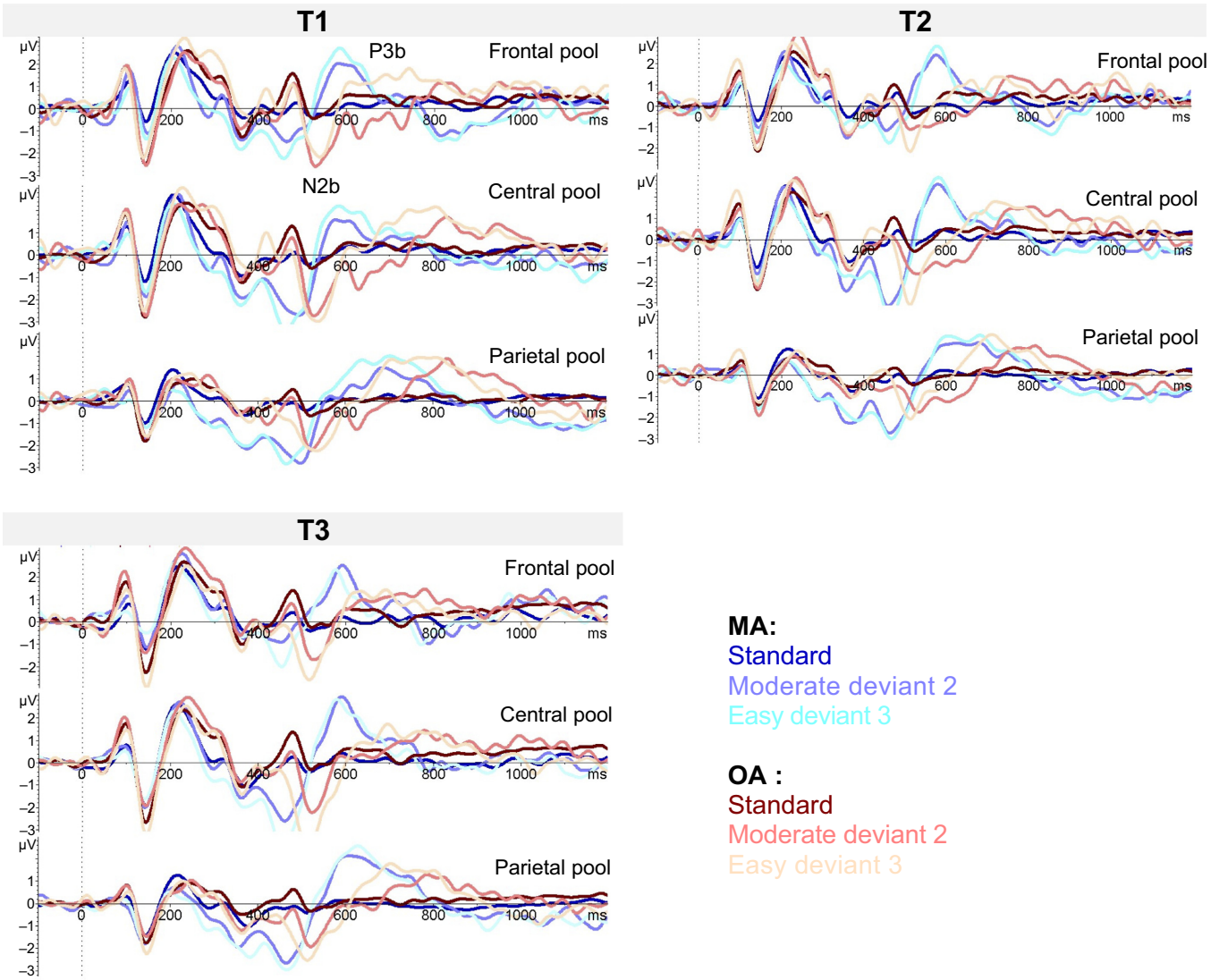

FIG. 2. The ERPs for three different electrode pools (frontal, central, and parietal) for the three measurement time points (T1, T2, T3) and for the standard stimulus, the moderate deviant, and the easy deviant, separately for the older adults (OA) in red tones and the middle-aged controls (MA) in blue tones. 0 ms on *x*-axis (time) represents syllable onset and 240 ms represents onset of spectral deviation. [Colour figure can be viewed at wileyonlinelibrary.com].

0.44 µV, MA: 0.49 µV). This finding implies that the posterior N2b was larger (as measured by deviant minus standard mean GFP) in MA than OA when evoked by the moderate deviant, but not the easy deviant. The T * STIM interaction ($P < .001$) further revealed that the mean GFP of Microstate 1 increased more strongly when evoked by the deviants compared to the standard stimulus. Furthermore, there was a T * STIM * group interaction ($P < .001$).

To sum up, the posterior N2b was larger and occurred with a shorter latency when evoked by the easier deviant compared to the more difficult deviant (the moderate deviant). The posterior N2b also occurred later and was smaller in OA compared to MA. Furthermore, the mean GFP of the posterior N2b increased with repeated exposure in both groups.

Microstate 2: central N2b

For the onset of Microstate 2, we did not find any significant results: [group ($P = .650$), T ($P = .128$), STIM ($P = .310$), T * group ($P = .101$), STIM * group ($P = .319$), T * STIM ($P = .054$), T * STIM * group ($P = .453$)]. Also, there was no main effect of group for the mean GFP of Microstate 2 ($P = .066$), no main effect T ($P = .873$), and no interaction between T * group ($P = .607$). However, the mean GFP of Microstate 2 changed as a function of STIM ($P < .001$), revealing that the easy deviant evoked higher mean GFP of Microstate 2 (0.63 µV) than the moderate deviant (0.51 µV) and the standard stimulus (0.19 µV). Furthermore, there was an interaction between STIM * group showing that in the OA group the deviants evoked lower mean GFP of Microstate 2 compared to the MA group (OA moderate deviant: 0.43 µV, MA moderate deviant: 0.76 µV, OA easy deviant: 0.57 µV, MA easy deviant: 0.87 µV), while the mean GFP evoked by the standard stimulus was not different between the two groups (OA: 0.21 µV, MA: 0.20 µV). This means that the central N2b was larger (as measured by deviant minus standard mean GFP) in MA compared to OA. Furthermore, the interaction between T * STIM ($P < .001$) revealed that the increase of mean GFP across T's was stronger for the easy deviant compared to the moderate deviant and the standard stimulus. Moreover, there was a threefold interaction of T * STIM * group ($P < .001$).

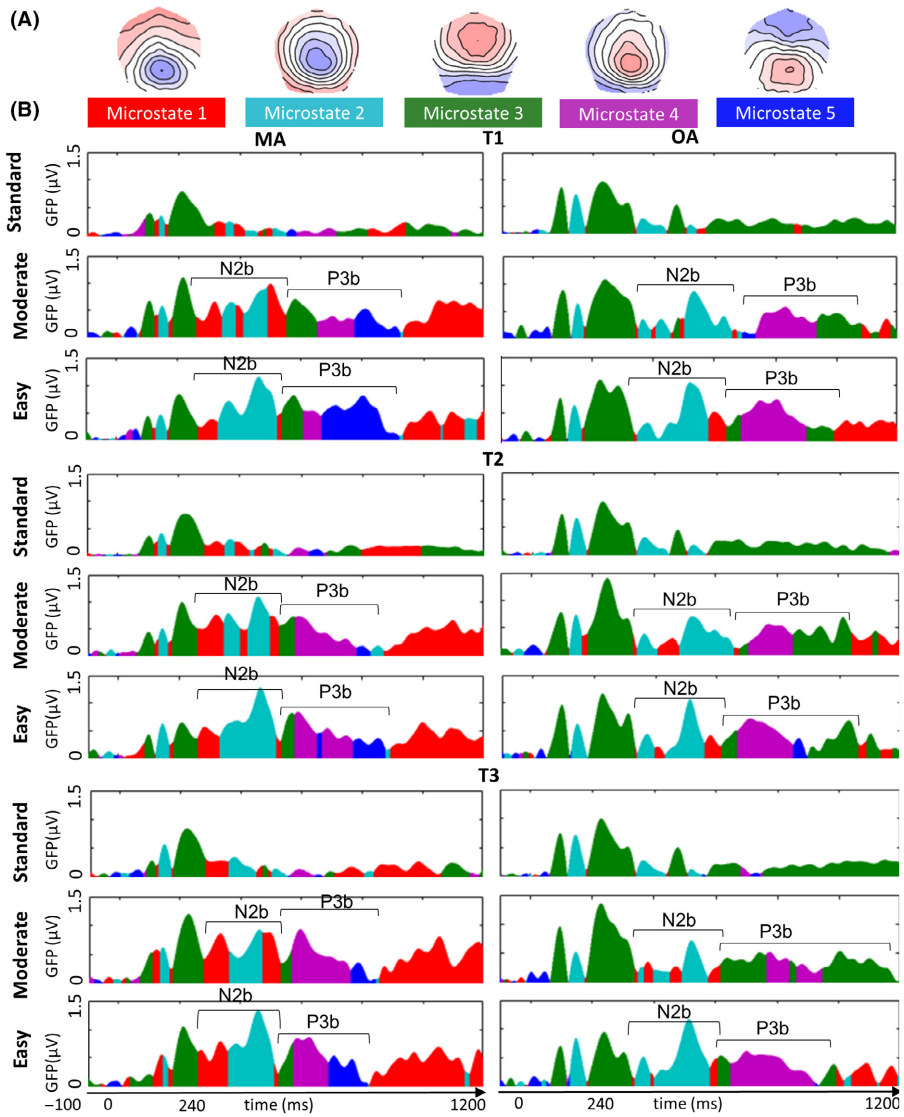

FIG. 3. (A) The results of the microstate segmentation for the EEG data consisting of the three measurement time points, the two groups, and the two deviants. It shows five stable topographical maps, Microstates 1–5 (color coded, orientation: nose up, left is left), which explain the EEG signal from −100–1200 ms after stimulus onset. (B) The time course of the mean GFP for each of the 5 microstates. OA, older adults; MA, middle-aged controls; T1, T2, and T3, measurement times points 1, 2, and 3; Standard, standard stimulus; moderate, deviant stimulus with weaker acoustic deviation from standard; easy, deviant stimulus with stronger acoustic deviation from standard. [Colour figure can be viewed at wileyonlinelibrary.com].

This means that the central N2b-related microstate (similar to the posterior N2b) was larger but did not show an earlier onset when evoked by the easier deviant compared to the more difficult deviant. In addition, and in a similar pattern found for the parietal N2b, the central N2b was smaller, but did not show a later onset in OA compared to MA. With repeated exposure, the mean GFP of the central N2b-related microstate increased especially when evoked by the easy deviant, irrespective of group.

Microstate 3: frontal P3b

The onset of Microstate 3 varied as a function of group (P = .034), T (P = .024), and STIM (P = .007). There were no significant interactions: [T * group (P = .255); STIM * group (P = .242); T * STIM (P = .284); T * STIM * group (P = .408)]. Microstate 3 occurred earlier in the MA group (540 ms) compared to the OA group (628 ms). Furthermore, the onset decreased from T1 to T2 (from 578 ms to 558 ms) in both groups. Moreover, irrespective of group, the onset was also different between STIMs, revealing that the onset was earlier when evoked by the standard stimulus (458 ms) as compared to when evoked by the deviants (both 564 ms).

For the mean GFP of the Microstate 3, we found a main effect STIM (P = .002), revealing that the mean GFP of Microstate 3 was lowest when evoked by the standard stimulus and highest when evoked by the easy deviant (standard: 0.18 μV, moderate deviant: 0.30 μV, easy deviant: 0.36 μV). Thus, the processing of the stimulus material resulted in a frontal P3b component, which was larger when evoked by the easy compared to the moderate deviant (when calculating deviant minus standard mean GFP). Furthermore, the analysis showed an interaction between STIM * T (P = .006), STIM * group (P < .001), and T * STIM * group (P < .001), while no main effect group (P = .066), no main effect T (P = .102), and no T * group interaction (P = .102) was found. The increase in mean

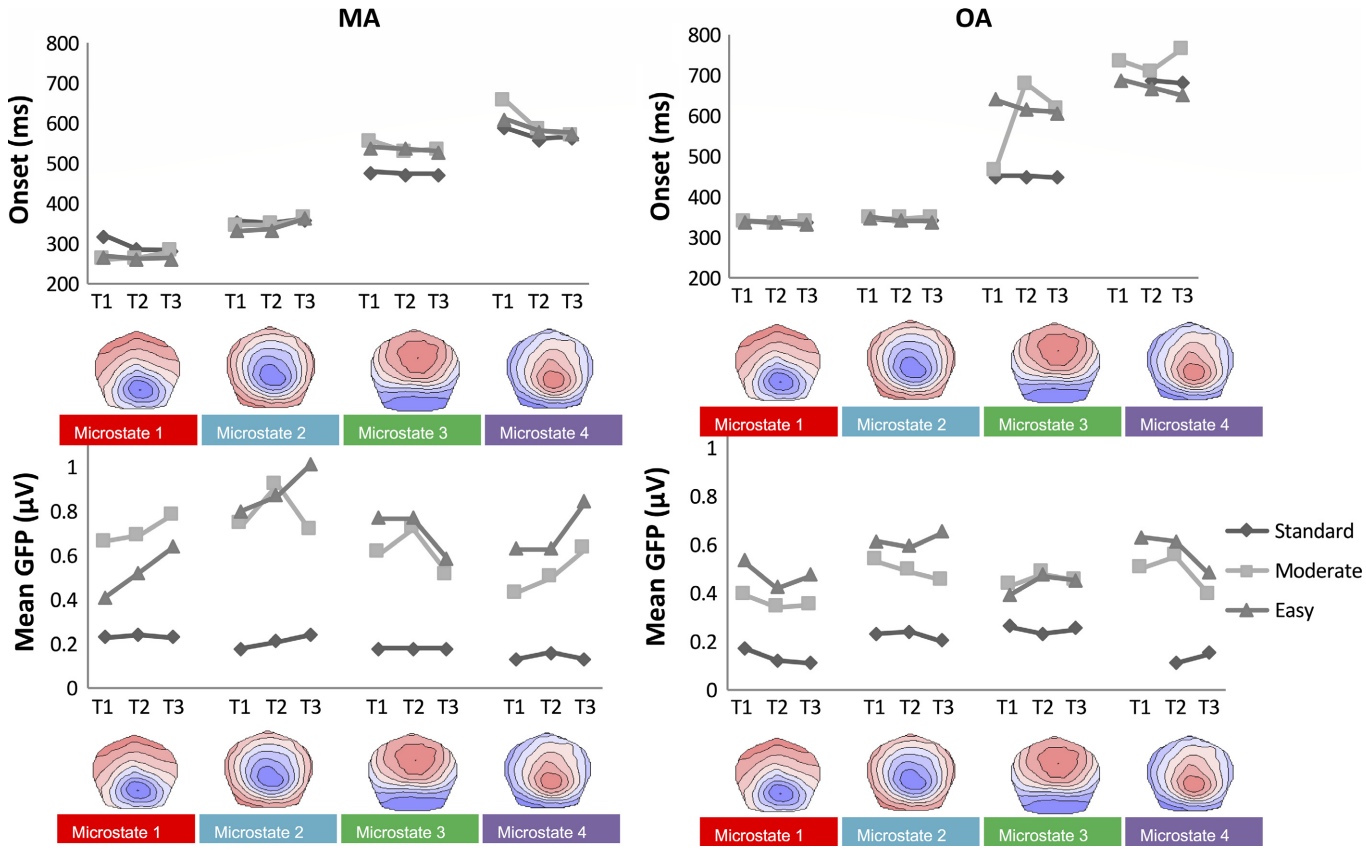

FIG. 4. The change of onset (above) and mean GFP (below) of four microstates (Microstates 1–4) across the three measurement time points (T1, T2, T3), separately for the middle-aged adults MA (left panel), and the older adults OA (right panel) for the standard stimulus (standard), the weaker deviant stimulus (moderate), and the stronger deviant stimulus (easy). Microstate 5 (posterior P3b-related microstate) is not depicted here because it was not evoked by the processing of the stimulus material in the OA group. [Colour figure can be viewed at wileyonlinelibrary.com].

GFP across T's was stronger for Microstate 3 when evoked by the moderate deviant as compared to the easy deviant. Furthermore, the mean GFP of Microstate 3 evoked by the deviants was lower in the MA group compared to the OA group, but the mean GFP of Microstate 3 evoked by the standard stimulus was higher in the OA group, leading to smaller frontal P3b-related microstates.

In sum, similar to the N2b-related microstates, the mean GFP of the frontal P3b was larger when evoked by the easier deviant compared to the more difficult deviant (the moderate deviant). In the OA group, the frontal P3b was smaller (similar to both N2b-related microstates) and had a longer latency (similar to the posterior N2b-related microstate) when compared to the MA group. Furthermore, irrespective of group, the onset decreased and the mean GFP increased with repeated stimulus exposure, especially when evoked by the more difficult deviant.

Microstate 4: central P3b

For the onset of Microstate 4, we found a main effect of group (P = .012) and a main effect of STIM (P = .046), revealing that Microstate 4 had an earlier onset in the MA group (586 ms on average) than in the OA group (688 ms on average) and that the onset was earlier in the easy deviant (608 ms on average) compared to the moderate deviant (632 ms on average). Furthermore, we found an interaction between T and STIM (P = .014), showing that the decrease in onset across T's was stronger when evoked by the moderate deviant compared to the easy deviant. There was no main effect of T (P = .083) and no interaction between T * group (P = .545). Notably, the interactions between STIM * group and T * STIM * group could not be computed because Microstate 4 did not occur in the standard stimulus of the OA group.

The mean GFP of Microstate 4 changed as a function of STIM (P < .001). The mean GFP was highest in the easy deviant condition and lowest for the standard stimulus (Standard: 0.11 µV, moderate: 0.28 µV, easy: 0.48 µV, on average). Furthermore, there was a significant interaction between T * STIM (P < .001), revealing that there was an increase in mean GFP in the moderate deviant, but not in the easy deviant or the standard stimulus across T's. There was no main effect of group (P = .674), no main effect of T (P = .205), and as mentioned above, the interactions between STIM * group and T * STIM * group could not be computed.

The central P3b (similar to the other microstates) was larger and occurred earlier when evoked by the easier deviant compared to the more difficult deviant. Furthermore, similar to the posterior N2b and the frontal P3b, the central P3b occurred earlier in MA compared to OA. However, this microstate was not larger in MA compared to OA. Similar to the frontal P3b, the mean GFP of the central P3b increased more strongly when evoked by the moderate compared to the easy deviant across measurement time points.

Microstate 5: posterior P3b

The posterior P3b-like microstate was only present in the MA group and not in the OA group (see Fig. 3B). It was therefore not possible

to calculate the group statistics. Thus, we refrained from further analyzing this microstate statistically.

Taken together, these results show that the better performance for the easier deviants was, as expected, accompanied by higher mean GFP and shorter onsets in all microstates. Furthermore, the age-related decline in behavioral spectral speech processing was, as predicted, reflected in lower mean GFP of the frontal P3b, the posterior N2b, and the central N2b, while the posterior P3b was completely missing in the older adults. Further, the age-related lower performance in the deviant detection task in OA was accompanied by a later onset of the N2b- and P3b-related microstates. In addition, we expected to find an increase in mean GFP and a decrease in onset of the microstates as a function of experience with the stimulus material. Thus, as predicted, with repeated stimulus exposure the mean GFP of the deviant stimuli increased in both groups, while the onset only decreased in the P3b-related microstates.

Discussion

Previous research has stated that speech processing relies on peripheral, central, and cognitive capacities (Humes *et al.*, 2012; Anderson *et al.*, 2013; Moore *et al.*, 2014; Füllgrabe *et al.*, 2015; Getzmann *et al.*, 2015). As cognition declines with age (Salthouse, 1996; Park & Reuter-Lorenz, 2009), older adults report that hearing leads to more effortful listening resulting in fatigue. However, increases in hearing loss originating in a decline in the auditory periphery and in central auditory circuits across the lifespan (Giroud *et al.*, in press) can be partially compensated for by cognitive abilities that may offset older adults' perceptual difficulties during speech processing (Kuchinsky *et al.*, 2016). Thus, the aim of the present study was twofold. First, it was of particular interest to better understand age-related differences in auditory neurocognitive speech processing. Second, a longitudinal design was used to evaluate to what extent familiarity with speech could facilitate cognitive processes, thus decreasing exhaustion during speech processing.

Using a repeated-measures design with an active oddball paradigm, the onset and the mean GFP of N2b and P3b-related microstates, signaling neurocognitive speech processes, were obtained for the processing of spectral information in high-pitched fricatives in normal hearing older adults and middle-aged controls. Our results showed that, irrespective of group, stronger acoustic deviation from the standard stimulus resulted in faster and higher accuracy in detection of this deviant, which is in line with our hypotheses (Johnson, 1986; Kok, 1997; Katayama & Polich, 1998; Gaál *et al.*, 2007; Giroud *et al.*, 2017b). This finding also conforms with the results of another study which showed that detecting small spectral differences in vowels resulted in longer reaction times and lower accuracy when compared to tasks where the spectral differences were more pronounced (Snyder & Alain, 2005). Interestingly, as indicated by the stimulus × group interaction, this stimulus effect was stronger in older adults; for the middle-aged controls, the two deviants were detected with a high accuracy already at T1 (MA: moderate deviant 0.98, easy deviant 0.99; OA: moderate deviant 0.83, easy deviant 0.92).

The finding that these behavioral differences between the deviants were reflected in electrophysiological correlates (Johnson, 1986; Kok, 1997; Katayama & Polich, 1998; Gaál *et al.*, 2007; Giroud *et al.*, 2017b) also confirmed our predictions. More precisely, we found lower mean GFP (for all microstates) and later onset (only in posterior N2b-related microstate and in central P3b-related microstate) for the deviants with weaker acoustic deviation to the standard. Such weaker acoustic differences have previously been related to higher task difficulty resulting in longer latencies (here, later onsets) in ERPs (Martin *et al.*, 2008; Giroud *et al.*, 2017b). Furthermore, the lower mean GFP evoked by the weaker deviants can be related to the lower attentional focus to that deviant, which is reflected by a smaller number of synchronously activated neurons in a wide-range bilateral frontal, parietal, limbic, cingulate, and temporo-occipital network (Volpe *et al.*, 2007).

As expected, we found age-related differences in the mean GFP and in the onset of the evoked microstates. Specifically, in the older compared to the middle-aged group, we found lower mean GFP and later onsets in N2b- and P3b-related microstates. Interestingly, it has previously been discussed that P3 amplitudes tend to be smaller in older adults during spectral processing (Bertoli *et al.*, 2005; Schiff *et al.*, 2008; Juckel *et al.*, 2012). The P3 has been previously associated with the anterior cingulate cortex, the superior temporal gyrus, and the fusiform gyrus, which are all parts of an attentional network (Juckel *et al.*, 2012). We therefore conclude that older adults probably perform worse in spectral processing, as reflected in lower GFP on the topography. This may be a result of lower neural responses in brain networks essential for both the categorization and the attentional processing of spectral acoustic details in an auditory stimulus. Furthermore, the activation of these networks appears to be slower in older adults, as evident in the later onsets of the N2b- and the P3b-related microstates. These longer latencies, however, do not seem to be specific to spectral processing, but rather to a general effect of aging on processing speed (Polich & Kok, 1995; van Dinteren *et al.*, 2014).

Are older adults more surprised than middle-aged controls when hearing an acoustic deviation? A discussion of the stronger frontal activation in older adults

The use of microstate analysis for assessing topographical distributions revealed further salient age-related differences, namely the posterior P3b-related microstate was only present in the middle-aged controls, and not in the older adults. In the older group, the deviants evoked more central and more frontal P3b-related microstates in the same ERP time window. These differences in frontal topographical distributions fit well into the accumulating evidence that cognitive processing in older adults is associated with stronger frontal activation (for overviews see Davis *et al.*, 2008; Reuter-Lorenz & Cappell, 2008; Reuter-Lorenz & Park, 2010). For example, within the posterior–anterior shift in aging (PASA) framework, the stronger activation in (pre)frontal areas in older adults is interpreted as a compensatory strategy against age-related neural decline, through the enlisting of additional top-down resources during memory tasks (Davis *et al.*, 2008). In addition, similar observations have been reported during speech processing tasks. To illustrate, during speech-in-noise word recognition, older adults showed reduced neural response in the bilateral posterior superior temporal gyri in comparison with younger adults, while the neural response was increased in the prefrontal and posterior parietal cortex at the same time (Wong *et al.*, 2009). Similarly, it has been reported that older adults show higher activation in frontal speech motor areas during syllable-identification-in-noise (Du *et al.*, 2016). However, the discussion on the nature of this stronger frontal activation is still ongoing. For example, it is still a matter of debate how frontal brain areas, which have been shown to be more strongly affected by age-related brain atrophy than sensory brain regions (Raz *et al.*, 1997; Tisserand *et al.*, 2002; Allen *et al.*, 2005; Abe *et al.*, 2008; Lemaitre *et al.*, 2012), are able to compensate for sensory decline. In addition, when investigating brain structural correlates of successful auditory performance in older adults, previous research has shown

positive correlations with cortical thickness in right auditory areas, such as the right planum temporale and the right Heschl's sulcus, rather than with frontal brain areas (Giroud *et al.*, in press).

In light of this debate, exploring alternative explanations for the higher occurrence of the P3b-related microstate with frontal activation on the scalp in older adults may be particularly advantageous. For example, previous research using a similar experimental design to ours, but with younger adults, also found a frontally distributed P3b-related microstate during active speech processing (Giroud *et al.*, 2017b). This microstate has been related to the P3a ERP component which is usually evoked during the processing of non-target stimuli, representing attentional allocation to surprising stimuli (Katayama & Polich, 1998). It can therefore be hypothesized that the deviant stimuli used in this experiment, although not new, may be surprising for a brief moment during processing, and that this is reflected in the occurrence of a frontal P300-related microstate (Giroud *et al.*, 2017b). Because this frontally distributed microstate is overrepresented in older adults compared to the middle-aged controls, it is possible that it flags a stronger surprise at the occurrence of deviant stimuli in older adults. Future research should test this hypothesis and investigate whether this stronger surprise in older adults toward deviating acoustic stimuli results from stronger decline in the memory trace for these deviant stimuli.

Auditory cognitive experience shapes how spectral stimuli are processed

We also predicted that, in the two groups, the neural representations of the stimulus material would change across measurement time points simply through the gaining of auditory experience, or mere repeated exposure to the fricatives (Tremblay *et al.*, 1998, 2010). In line with this prediction, we found that the reaction times decreased from T1 to T2 for the oddball task and were flagged by a decrease in the onset of the P3b-related microstates. However, previous studies investigating longitudinal changes in earlier ERP components like the N1 and the P2 have not found any changes in the latency through repeated exposure (Menning *et al.*, 2000; Brattico *et al.*, 2003; Bosnyak *et al.*, 2004; Wagner *et al.*, 2013; Tremblay *et al.*, 2014). One exception is a study which also investigated spectral processing using a vowel identification task (Reinke *et al.*, 2003). In our study, the decreases in onset across the measurement time points were specific to the P3b-related microstate. This finding shows that mainly the attentional and memory updating processes were executed faster with increasing familiarity with the stimulus material (Giroud *et al.*, 2017b). Moreover, the mean GFP of all microstates increased across the three measurement time points, but this mainly for the deviants and not the standard. This shows that the N2b- and the P3b-related microstates, when defined as the difference between the mean GFP of the deviants minus the mean GFP of the standard, increased with repeated exposure. By consolidating and storing the stimulus material in long-term memory between the measurement time points (Karni & Sagi, 1993; Atienza *et al.*, 2002), the spectral differences between the deviants and the standard become more salient, which was reflected in the increasing N2b and P3b responses across the measurement time points.

Limitations and outlook

This research has some limitations to be discussed. First, in order to investigate more difficult tasks, such as using deviants with weak acoustic deviation from the standard, more trials need to be introduced to the paradigm. However, gaining sufficient correct trials for difficult tasks from each participant also means that the EEG data acquisition will increase in time, which is not suitable for older adults. Thus, for this research, we decided to keep the EEG data acquisition below one hour and therefore only used deviants which can be easily detected by all participants.

Also, it would be interesting to use a complementary strategy for age group comparisons. Investigating individual differences within each age group such as the influence of biographical data like musical experience (Parbery-Clark *et al.*, 2012a,b), peripheral and central hearing using a comprehensive test battery (Giroud *et al.*, in press), cortical atrophy and other structural brain measures (Giroud *et al.*, in press), and individual cognitive capacities (Arlinger *et al.*, 2009) on auditory cognitive learning could provide more comprehensive and individualized models on how to support speech processing in older adults.

Conclusion

We conclude that there is a transition in cognitive auditory hearing functions from middle age to old age in that older adults perform worse in spectral discrimination because of lower and slower activation in the underlying brain networks which mediate auditory neurocognitive processes. Furthermore, our data provide novel evidence from the domain of hearing research for the neurocognitive aging model PASA, which predicts stronger frontal activation in older adults during sensory-cognitive tasks (Davis *et al.*, 2008). However, because the model still lacks support from neurostructural evidence (Giroud *et al.*, in press), alternative explanations for the stronger frontal activation in the older group may be more suitable. For example, our data suggest that the frontal P300-related microstate relates to surprise of perceived acoustic deviation, thus promoting the allocation of attention to the stimulus (Giroud *et al.*, 2017b). This process seems to be stronger in older adults compared to middle-aged controls.

Furthermore, our longitudinal approach revealed that both age groups benefitted from familiarity with the stimulus material due to repeated exposure, as evident in the decrease of the reaction times for detecting acoustic deviations across measurement time points. This behavioral plasticity was further accompanied by increasing brain responses in the time windows of the N2b- and the P3b-related microstates, and also by a decrease of the latency at which the attention was drawn to spectral acoustic deviations in the stimuli. In sum, these results suggest novel evidence that, with increasing familiarity with the stimulus material through repeated exposure, neurocognitive speech processing can be facilitated in middle-aged and older adults.

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Conflict of interest

The authors have declared that no competing interests exist.

Author contributions

UL, PR, MM, and NG contributed to the conception and design of this study. PR, SW, JB, and NG collected the data and JB and NG analyzed the data. NG drafted the manuscript, and UL, MM, JB,

and SW were involved in revising the article. All authors have approved the final version of the manuscript to be published.

Data accessibility

The data used in this study are not accessible, because participants did not give consent that their data are available beyond this study.

Abbreviations

ACC, accuracy; ERP, event-related potential; GFP, global field power; MA, middle-aged adults; OA, older adults; RT, reaction time; SNR, signal-to-noise ratio; STIM, stimulus (standard, moderate deviant, easy deviant); *T*, measurement time point (T1, T2, T3).

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